EXHIBIT C

UNITED STATES DISTRICT COURT
FOR THE MIDDLE DISTRICT OF NORTH CAROLINA
GREENSBORO DIVISION

Lloyd Bell, Individually and as Executor of the Estate of Betty Whitney Bell, Deceased	:	
Plaintiffs,	:	File No.: 1:17-cv-00111
vs.	:	
American International Industries, Inc. et al.	:	
Defendants.	:	

DECLARATION OF JACQUELINE MOLINE, MD, MSc, FACP, FACOEM

JACQUELINE MOLINE, MD, MSc, FACP, FACOEM, hereby declares under penalty of perjury the following:

1. My name is Dr. Jacqueline Moline. I am a physician licensed in the State of New York, specializing in the field of occupational and environmental disease. I have been a practicing physician since I graduated from medical school at the University of Chicago – Pritzker School of Medicine in 1988.

2. Following medical school graduation, I was an intern and resident in Internal Medicine at Yale University, Yale New Haven Hospital, from 1988-1991. Upon completion of my Internal Medicine Residency program, I completed a second residency at the Mount Sinai School of Medicine in Occupational Medicine, from 1991-1993. During my Occupational Medicine Residency Program, I obtained my Master of Science Degree in Community Medicine (equivalent degree to a Masters of Public Health) in 1993. I began to evaluate dozens of patients with asbestos exposure during my residency program at Mount Sinai. I am board certified in Occupational Medicine and in Internal Medicine. I have become recertified in Internal Medicine two times. I currently direct the Occupational and Environmental Medicine Center of Long Island, providing occupational health services to patients in the metropolitan New York area.

3. I have evaluated hundreds of patients with asbestos exposure in my career in occupational medicine, spanning nearly 30 years. Over the past year alone, I have supervised the examination of or directly examined nearly 500 patients with asbestos exposure. Over the course of the past years, I have evaluated dozens of patients with malignant mesothelioma and lung cancer due to asbestos exposure. I have kept abreast of the scientific and medical literature regarding the diagnosis and causation of mesothelioma. I have personally evaluated cases of mesothelioma where the exposure was brief, and have also seen cases of mesothelioma in individuals whose only exposure to asbestos was from family members who worked with asbestos and brought their asbestos contaminated clothes home. I have published in the peer-reviewed literature on occupational and environmental diseases, including mesothelioma.

4. I have previously been retained and have testified as an expert witness in litigation in state and federal court cases involving asbestos exposure and disease causation, including matters involving exposure to asbestos-containing talc and consumer talcum powder products causing mesothelioma. My methodology was subject to a Daubert hearing and has been found reliable. My opinions regarding asbestos-containing talc and its ability to cause mesothelioma have been upheld on appeal. A true and correct copy of my curriculum vitae is attached hereto as **Exhibit A**.

Summary of Opinions and Specific Causation

5. I have been retained as an expert in this case for my expertise in asbestos exposure and disease causation. I have had the opportunity to review materials including the depositions of decedent Betty Bell, Ms. Bell's late husband Lloyd Bell, as well as Donald Bell, Judy Drye, Nancy Taylor, and Wayne Whitley, medical records and pathology reports of Betty Bell, the depositions of the corporate representatives for various cosmetic talc companies including but not limited to Colgate-Palmolive, American International Industries for Clubman, Avon, Johnson & Johnson, the reports of material scientists and microscopists William Longo, PhD,[1] Steven Compton, PhD,[2] and Sean Fitzgerald,[3] the reports of statistician David Madigan, PhD,[4] company records of cosmetic talcum powder product manufacturers and their suppliers, including but not limited to Colgate-Palmolive Company, Johnson & Johnson, Imerys/Cyprus, and Whittaker Clark & Daniels, trade organization documents, articles in the medical and scientific literature, government records, and other studies relevant to asbestos-containing talc and consumer talcum powder products including historical productions of Cashmere Bouquet and Clubman, and the source ores of each, to determine if it was my opinion, to a reasonable degree of medical certainty, that Betty Bell's use of and exposure to asbestos-containing Cashmere Bouquet and Clubman, would have resulted in a significant exposure to asbestos and would have been a substantial factor in causing her mesothelioma and resulting death.

6. Based on the information that was provided to me, and applying both my understanding of the medical literature and the facts of this case, it is my opinion to a reasonable degree of medical certainty that the exposures to asbestos from Cashmere Bouquet and Clubman

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[1] A true and correct copy of the Declaration of William Longo, PhD is attached hereto as **Exhibit B**. The attachments to Dr. Longo's Declaration have not been included due to volume. They will be provided upon request. The laboratory reports generated by Dr. Longo for his testing of Cashmere Bouquet are voluminous. I have reviewed all of the underlying reports that are summarized in Exhibit B but have not attached them to this declaration. I maintain copies of each of the reports and can provide them if necessary.

[2] A true and correct copy of the Declaration of Steven Compton, PhD in this case, is attached hereto as **Exhibit C**. The attachments to Dr. Compton's Declaration have not been attached due to volume. They will be provided upon request. The laboratory reports generated by Dr. Compton for his testing of Italian talc, Montana talc, Clubman, and Cashmere Bouquet are voluminous. I have reviewed all of the underlying reports that are summarized in Exhibit C but have not attached them to this declaration. I maintain copies of each of the reports and can provide them if necessary.

[3] A true and correct copy of the report of Sean Fitzgerald, P.G. of SAI, Inc. in this case is attached hereto as **Exhibit D**. The attachments to Mr. Fitzgerald's report have not been attached due to volume. They will be provided upon request.

[4] A true and correct copy of the reports of David Madigan, PhD dated May 6, 2019 and June 26, 2019, and is attached hereto as **Exhibit E**. The attachments to Dr. Madigan's reports have not been attached due to volume. They will be provided upon request.

talc that Betty Bell used and was exposed to for almost five decades, beginning in the 1950s and continuing through 2009, were above normal background levels.

7. Both historic and recent analyses (published in the medical and scientific literature as well as industry, government and private laboratory testing) of the talc from the source mines used in Cashmere Bouquet and Clubman,[5] as well as of Cashmere Bouquet and Clubman finished powder products, have shown significant amounts of chrysotile, anthophyllite and tremolite asbestos. Studies done recently from products using ore taken from the same source mines as those used in the manufacture of the Cashmere Bouquet and Clubman, showed significant amounts of chrysotile, anthophyllite, and tremolite asbestos. Dr. Steven Compton found asbestos (anthophyllite, tremolite, actinolite, and chrysotile) in 11 samples collected from the Italian mining region from which the talc originated that was then used in consumer products, including Cashmere Bouquet and Clubman. (Exhibit C) In 1984, MSHA found anthophyllite, this same fiber type found by Dr. Compton, in the mill that processed the Italian talc.[6] In fact, based on MSHA's findings, it was estimated that 0.6% of the Italian talc sold by Cyprus/Imerys to companies for use in consumer talcum powder products is anthophyllite asbestos.

8. The asbestos found in the ores is also found in the finished consumer products.[7] Dr. William Longo found tremolite and anthophyllite in his analysis of historic Cashmere Bouquet powder products, the same fiber types found in the historic testing records of Italian, North Carolina,

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[5] Deposition of John Hopkins, PhD, corporate representative of Johnson & Johnson, August 15-18, 2017 and exhibits thereto; March 25, 1992 Excerpts from Cyprus Talc Reserve Report by R.C. Munro; August 28, 1973 – Johns Manville TEM talc testing/14 samples. Memo to R.S. Lamar from V.E. Wolkodoff, Subject: "Determination of Chrysotile by TEM in Commercial Talcs."; December 10, 1973 – Round Robin test of Italian, Montana, Alabama, and North Carolina talc. "Report of CTFA Method to Detect Chrysotile and Tremolite in Talc"; 1972 – Investigation of Possible Asbestos Contamination in Talc Samples, University of Minnesota Space Science Center; Rohl, A., Langer, A., et al., Consumer Talcums and Powders: Mineral and Chemical Characterization, Journal of Toxicology and Environmental Health, 2:255-284 (1976); Summary of TEM Asbestos Results: Grade 66/96 USP Product Composites (March 11, 2004); October 31, 1968 Johns-Manville testing, Body Talcum Powders – Petrographic Examination; Report from Lewin to Weissler of FDA dated August 3, 1972; Memorandum from Weissler of FDA re Summary and Comments on Prof. Lewin's Analytical Results for Asbestos in Talc, July 31, 1973; Report from Lewin to WCD re 1615 Talc Analysis, September 30, 1972; Windsor Minerals New Reagents Systems report Cyprus, May 14, 1974; Windsor Minerals Corporation Samples to McCrone and corresponding results identifying anthophyllite, November 2, 1990.

[6] May 15, 1984 – Report of MSHA visit to Cyprus Industrial Minerals Company South Plainfield Mill.

[7] Deposition of John Hopkins, PhD, corporate representative of Johnson & Johnson, August 15-18, 2017 and exhibits thereto; Deposition of Lisa Gallo, corporate representative of Avon, and exhibits thereto; 1972 – Investigation of Possible Asbestos Contamination in Talc Samples, University of Minnesota Space Science Center; Rohl, A., Langer, A., et al., Consumer Talcums and Powders: Mineral and Chemical Characterization, Journal of Toxicology and Environmental Health, 2:255-284 (1976); Report from Lewin to Weissler of FDA dated August 3, 1972; Memorandum from Weissler of FDA re Summary and Comments on Prof. Lewin's Analytical Results for Asbestos in Talc, July 31, 1973; Inter-Office Correspondence from Sloan of Johnson & Johnson re Telephone Conversation – Dr. F. Pooley – March 31, 1976; Bowes, Report of the Examination of American Consumer Talc Samples; RJ Lee Group letter report to Ashton re-examination of three talcum powders detecting tremolite in Johnson's Baby Powder, October 13, 1995; Forensic Analytical Report on JBP, February 24, 2004; AMA Analytical Services, Inc. Certificate of Analysis for US Food & Drug Administration (Johnson's Baby Powder Lot #22318RB), October 11, 2019.

and Montana talc ores and in historic testing records of Cashmere Bouquet. (Exhibit B) Dr. Longo analyzed and reported on approximately 58 Cashmere Bouquet products that were manufactured from 1910 through 1977 and provided to his laboratory from mesothelioma plaintiffs, from collectors, and from Colgate-Palmolive's own historic collection (20 obtained directly from Colgate-Palmolive). (Exhibit B) Dr. Longo reported regulated asbestos in 51 of 58 (88%) containers of Cashmere Bouquet talcum powder products. (Exhibit B).

9. Three independent laboratories have identified asbestos in Betty Bell's own container of Clubman.[8] Mr. Fitzgerald of SAI, Inc. and Dr. Compton of MVA both identified tremolite asbestos in the Clubman container Ms. Bell found in her home and gave to her lawyers. (Exhibit C, Exhibit D) The vintage of Ms. Bell's container was one in which Italian talc was used to manufacture the Clubman.[9] As noted above, tremolite has been repeatedly identified in Italian talc. Dr. Compton has also identified amphibole asbestos in three containers of Clubman manufactured with Italian and Montana talc as well as a barber brush containing Clubman talc. (Exhibit C)

10. Betty Bell's use of and exposure to asbestos-containing Cashmere Bouquet and Clubman talcum powder products was a substantial factor in causing her mesothelioma and subsequent death. Alternative powders that did not contain talc (and therefore did not contain asbestos) were available since the early 20th century.[10]

Betty Bell's Medical and Exposure History

Clinical History:

11. Ms. Bell was a 66 year old woman who was in her usual state until March 2015 when she developed shortness of breath, chest wall pain and fatigue. She saw Dr. McCollough, her primary care provider, on March 20, 2015, and was started on diuretics along with anti- anxiety and anti-depressant medication. An echocardiogram on April 1, 2015 showed normal left ventricular size and function with an ejection fraction of 55%. There was mild diastolic dysfunction.

12. Ms. Bell returned to Dr. McCollough on April 27, 2015 and noted persistent shortness of breath, right upper quadrant pain and fatigue. She had left knee and low back pain that had not been relieved by tramadol. She had been started on Zoloft and Clonazepam for anxiety

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[8] SAI, Inc. of Greensboro, North Carolina, Lab/Cor of Seattle, Washington, and MVA Scientific Consultants of Duluth, Georgia.

[9] In 1987, American International Industries (AII) purchased the exclusive rights to the Clubman brand group of products, including Clubman talc from The Neslemur Company. *See* Deposition of Charlie Loveless, August 12, 2016. At that time, AII switched from Italian talc to Montana talc. *See* Deposition of Theodore Hubbard, August 8, 2016. The container of Clubman Ms. Bell still had in her possession was metal and therefore manufactured before the early 1990s. *See* Deposition of Charlie Loveless, August 12, 2016.

[10] July 13, 1966 Johnson & Johnson document Bates Numbers JNJAZ55_000000049-JNJAZ55_000000051; *see also* testimony of Johnson & Johnson's corporate representative John Hopkins, Ph.D. According to Colgate, corn or rice starch has been used as a substitute for talc as a dusting powder since the mid-1950s (approximately 1954) due to talc's low absorption rate and irritating effect on the skin of babies. *See* U.S. Pat. No. 4,407,798 (Oct. 4, 1983) filed by Colgate-Palmolive Company.

without significant relief. Dr. McCollough ordered a right upper quadrant ultrasound that was done on May 6, 2015 and showed gallstones and a right pleural effusion. Ms. Bell had a chest x-ray on May 27, 2015 that showed a small to moderate right pleural effusion with associated lower lobe atelectasis or infiltrate. Dr. McCollough saw her on May 27th and noted that she was still short of breath. She had recently had a steroid injection in her right knee for osteoarthritis. Dr. McCollough referred her to interventional radiology for a thoracentesis, and also treated her for anxiety and depression.

13. She underwent a thoracentesis on June 1, 2015 under ultrasound guidance. About 1,600 cubic centimeters of fluid was removed. The pleural fluid had malignant cells with non-small cell carcinoma of the lung favored. Dr. McCollough referred her to hematology/oncology for further management and evaluation. Dr. Thomas Steffens, a medical oncologist, saw Ms. Bell on June 9, 2015. Ms. Bell had fatigue and shortness of breath with exertion; she had difficulty swallowing at times. She had lost 25 pounds without trying to lose weight in the past three months and had mild sweats/hot flashes. Dr. Steffens noted that she had a malignant effusion, but the etiology was unclear. Because Ms. Bell was having headaches, he ordered an MRI of the brain and a PET/CT scan. He also discussed the need for additional tissue to make a diagnosis. She was also seen by social work because of an increased distress scale.

14. Ms. Bell underwent a PET/CT scan on June 10, 2015 that showed no abnormal FDG uptake involving the thorax, abdomen or pelvis. A brain MRI was negative, and a mammogram was negative.

15. A repeat thoracentesis was done on June 16, 2015 with about 500 cubic centimeters of fluid removed. The cytology showed abnormal cells consistent with mesothelioma. A post-procedure chest x-ray showed no residual right pleural effusion. Ms. Bell returned to Dr. McCollough on June 25, 2015. She was still short of breath and had chest discomfort. She also had back and knee pain.

16. Dr. Thomas D'Amico saw Ms. Bell for an initial evaluation on July 7, 2015. Ms. Bell had pain beneath her right breast and shortness of breath with cough. She had lost around twenty pounds and had early satiety. Dr. D'Amico noted that Ms. Bell was in need of a tissue diagnosis, and planned a right thoracoscopic biopsy and pleurodesis. He noted that she had worked for Philip Morris Tobacco for 12 years, and had significant second hand smoke exposure. In addition, she had worked in a cotton mill and in the fiber industry for many years starting in the 1960s. A chest CT scan on July 7, 2015 showed a moderate to large pleural effusion with mediastinal pleural thickening at the level of the superior vena cava-ascending aorta. There was partial atelectasis of the medial segment of the right lower lobe. Her kidneys had an irregular contour. There was mild bronchial wall thickening in the left lung.

17. Ms. Bell was admitted to Duke University Medical Center on July 20, 2015. Dr. D'Amico performed a thoracoscopy and pleural biopsy, with talc pleurodesis. At the time of surgery, he noted a moderate pleural effusion, and removed around 500 cubic centimeters of fluid. There were pleural nodules in the anterior mediastinal pleura, parasternal pleura, and on the thoracic pleura near the diaphragm. The pathology showed malignant mesothelioma, epithelial variant, in the pleura and subpleural fibromuscular tissue. A hyaline pleural plaque was also noted. There were no complications following surgery, and Ms. Bell was discharged home on July 22, 2015.

18. Ms. Bell went to see Dr. Frank Dunphy II, a medical oncologist, on August 11, 2015. She was no longer short of breath. Ms. Bell's tumor was staged as T1bN0M0. He planned three courses of chemotherapy with Pemetrexed and Cisplatin, followed by a repeat CT scan four weeks after the third chemotherapy cycle to see if she was a surgical candidate for a right pleurectomy. He ordered a swallowing study because of Ms. Bell's symptoms of dysphagia and her history of prior esophageal dilatation. A chest x-ray showed decreasing air in the right apical pleural space with minimal basilar partial atelectasis and possible tiny amount of right sided pleural fluid or thickening. Dr. D'Amico also saw Ms. Bell on August 11th. She was feeling well since surgery, and he noted that chemotherapy was scheduled to start on August 13, 2015.

19. Ms. Bell received her first cycle of chemotherapy on August 13, 2015. She returned to Dr. Dunphy's office on August 18, 2015. She had noted some heart pounding and mild nausea. Her platelet count was low at 79,000. She was advised to increase her fluid intake. Ms. Bell returned to Dr. Dunphy on September 1, 2015 for her second cycle of chemotherapy. She had a videofluoroscopic swallow study on September 2, 2015. Ms. Bell was noting difficulty with solids, with a sensation that food was getting stuck. Her swallowing study showed normal oropharyngeal swallow function. A cardiac echo was done on September 2, 2015. There was normal left ventricular systolic function with mild left ventricular hypertrophy.

20. Ms. Bell developed a fever with chills, nasal congestion and laryngitis on September 8, 2015. She was hospitalized at North East Medical Center with dehydration, neutropenia and thrombocytopenia, and started on antibiotics and intravenous fluids. Her hemoglobin was only 7.1 grams and her platelet count was 13,000. Ms. Bell received a transfusion of packed red blood cells and platelets on September 12, 2015. She was also given magnesium and potassium supplementation. During the hospitalization, Ms. Bell was hoarse, which was felt to be related to a viral illness. She completed a course of antibiotics and was discharged home on September 17, 2015.

21. Ms. Bell returned to Dr. McCollough on September 24, 2015. She was fatigued and was unable to take the potassium supplements because the tablets were too big and caused her to vomit. She had lost twenty pounds since April. Her magnesium and potassium levels were still low. She was still hoarse, and Dr. McCollough planned to refer her to an ear, nose and throat specialist if the hoarseness did not improve. NP Yeshu Conn from Dr. Dunphy's office saw Ms. Bell on September 30, 2015. She was still hoarse and fatigued. She was switched from Cisplatin to Carboplatin and her dose of Pemetrexed was reduced as well. She received Neulasta on September 30th.

22. Dr. Seth Cohen saw Ms. Bell on October 2, 2015 because of persistent hoarseness, which Ms. Bell noted had started three days after chemotherapy started. Her voice was still weak and breathy. Dr. Cohen performed a laryngoscopy and noted that there was bilateral vocal fold erythema with ulcerative lesions with supraglottic hyperfunction and incomplete closure, along with muscle tension dysphonia. He prescribed a short course of steroids and voice rest to reduce the inflammation.

23. Ms. Bell returned to Dr. Dunphy's office on October 6, 2015. She had new mild right upper quadrant and upper abdominal pain that had been present for the prior week. She was on antibiotics as prophylaxis for infection. NP Yeshu Conn ordered an abdominal CT scan because of a concern about diaphragm involvement of cancer. Ms. Bell developed severe weakness and did not even have the energy to shower and her limbs felt weak and heavy like she "couldn't lift them". She

went to Dr. Steffens office and was seen by PA Sarah Brandt, who found that Ms. Bell had severe anemia with a hemoglobin of 6 grams. A chest x-ray showed a minimal right pleural effusion and chronic pleural fissural thickening. She was treated with three units of packed red blood cells on October 15th and 16th, 2015. Ms. Bell was encouraged to increase her oral intake and drink at least three supplements a day. She was scheduled to see a gastroenterologist for the abdominal pain.

24. Dr. Cohen saw Ms. Bell on October 19th. A videostroboscopy was performed and showed persistent but improved vocal fold erythema with smaller ulcerative lesions and incomplete but improved closure of the vocal cords. There was a marked decrease but improved periodic vibration of the vocal cords. Dr. Cohen planned to see her in 4-6 weeks. A CT scan of the abdomen and pelvis on October 19th showed no definite acute abnormalities in the abdomen or pelvis. There was a moderate right pleural effusion that was slightly decreased in size when compared to prior CT scans. There were gallstones without evidence of cholecystitis.

25. Ms. Bell returned to Dr. D'Amico for consideration for additional surgery. She was feeling better and had more energy and had been able to increase her activity levels. Pulmonary function tests on November 10, 2015 showed a forced vital capacity of 2.61 liters (81% of predicted) and a forced expiratory volume in the first second of 1.86 liters (76% of predicted). The diffusion capacity was only 56%. A CT scan of the chest, abdomen and pelvis on November 10, 2015 showed no evidence of metastatic disease in the abdomen. There were degenerative changes seen and some atherosclerotic calcifications.

26. Ms. Bell underwent a decortication and parietal pleurectomy with resection of the diaphragm with a mesh/flap and resection of the pericardial tumor on December 5, 2015. She required blood pressure support after surgery and received blood transfusions for anemia. The pathology showed minute foci of residual malignant mesothelioma in the parietal pleura in a background of extensive talc pleurodesis. There was invasion of the mesothelioma into the adipose tissue. Ms. Bell had ongoing right upper quadrant pain, nausea, vomiting, fevers and tachycardia and had decreased blood pressure. She was transferred back to the intensive care unit on December 11, 2015 and received blood transfusions. A CT scan of the chest showed an interval increase in the right pleural fluid with multiple internal foci of air, concerning for a hematoma. There was mediastinal pericardial stranding with hemorrhagic collection in the right medial pleural pericardial region. There was a large hematoma in the right posterior chest wall. A small pneumothorax was seen. A CT scan of her abdomen was unremarkable apart from possible stranding in the kidney. She had an increase in her serum creatinine. An abdominal ultrasound showed multiple gallstones with trace pericholecystic fluid, and was indeterminate for acute cholecystitis. She was treated with antibiotics. A HIDA scan of the gallbladder showed no definite evidence of acute cholecystitis. Blood cultures were positive for bacteria. Ms. Bell continued to require blood and platelet transfusions. A CT scan of the chest on December 16, 2015 showed an interval increase in the loculated right hemithorax. There was an unchanged anterior mediastinal hematoma and right chest posterior wall hematoma. Ms. Bell slowly recovered and was discharged home on December 22, 2015.

27. Dr. McCollough saw Ms. Bell on March 31, 2016. She was on surveillance. Dr. McCollough increased her anti-depressant medication, since she was now also anxious and depressed due to her husband's recent diagnosis of non-Hodgkin's lymphoma. Dr. D'Amico saw Ms. Bell on May 3, 2016. She noted no significant change in her symptoms. She was taking Tramadol, but it was of limited effect. A CT scan on May 3, 2016 showed resection of the previously noted pleural masses. There were scattered areas of mild pleural thickening and pleural nodularity.

There was a small amount of right pleural fluid. There was mild asymmetric thickening of the right lateral chest wall that could be post-operative or potentially from chest wall tumor invasion. Dr. D'Amico started Ms. Bell on Gabapentin and planned to see her in three months.

28. Ms. Bell returned to Dr. D'Amico on November 8, 2016. She noted slowly worsening shortness of breath and a dry cough for the prior month. She had pain in her right chest wall, mostly under her right breast but above as well and wrapping around her side. A CT scan on November 8, 2016 showed interval increase in the soft tissue thickening and nodularity in the right pleural space, likely representing worsening of disease. There was soft tissue thickening of the right lateral chest wall as well. Dr. D'Amico was concerned about disease progression, and referred her to Dr. Thomas Stinchcombe for further oncologic treatment.

29. Ms. Bell went to Dr. Stinchcombe on December 8, 2016. He noted she received three cycles of Cisplatin and Pemetrexed prior to surgery with complications requiring surgery. He planned two cycles of Carboplatin and Pemetrexed, starting on December 29, 2016. A repeat CT scan on December 29, 2016 showed pleural nodules and masses, slightly increased from November 2016. There was a right lateral chest wall soft tissue opacity that infiltrated the lateral chest wall musculature. A possible enlarging subcarinal lymph node was noted. Ms. Bell received chemotherapy on December 29th, 2016. A repeat CT scan on February 1, 2017 showed a slight decrease in the degree of pleural nodularity. Ms. Bell continued to receive chemotherapy every three weeks, complicated by anemia requiring transfusions. Ms. Bell had a CT scan of the head on March 24, 2017 after suffering from dizziness. There was no acute intracranial abnormality. A CT scan of the chest on March 24, 2017 showed stability to slight interval decrease in the extent of the right pleural mesothelioma. There was no further disease progression. Ms. Bell was admitted to the hospital with a neutropenic fever, fatigue and nausea on March 27, 2017. She received a platelet transfusion and became febrile. She was also dehydrated. She was treated with fluids, antibiotics and a blood transfusion for a hemoglobin of 5.8 grams. She was profoundly thrombocytopenic and required platelet transfusions. Ms. Bell was discharged on April 2, 2017, with plans to increase the time interval between chemotherapy to allow for her bone marrow to recover.

30. A bone marrow biopsy was done on April 6, 2017. There was a hypocellular marrow with a decreased number of megakaryocytes. Ms. Bell was admitted to the hospital on May 18, 2017 with nausea, vomiting, diarrhea, weakness, dehydration and slight abdominal discomfort. An abdominal CT scan showed progression of the right pleural mesothelioma with multiple liver metastases and omental caking suspicious for malignant involvement of the omentum. She was given intravenous fluids, blood transfusions and platelet transfusions. She was discharged home on May 21, 2017. There was discussion of possible liver biopsy.

31. Ms. Bell developed further weakness, nausea, vomiting and had poor oral intake at the end of May 2017. She was admitted to the hospital on May 30, 2017 with low blood pressure and a very low temperature. She was treated for sepsis and given fluids and antibiotics. A CT scan of the abdomen showed extensive peritoneal carcinomatosis with significant interval increase in the volume of ascites. There was hepatic metastatic disease and extensive pleural based masses and nodularity compatible with the known mesothelioma. There was a small left pleural effusion. An abdominal ultrasound on May 31, 2017 showed metastatic neoplastic lesions in the liver. There was gallbladder sludge and gallstones. There was ascites consistent with known peritoneal carcinomatosis. Ms. Bell developed more abdominal pain. She was placed on hospice care on June 1, 2017.

32. Ms. Bell died on June 3, 2017. She was 67 years old.

Past Medical History

33. Ms. Bell had a history of arthritis, gastroesophageal reflux disease, and esophageal stricture. She had a hysterectomy. Ms. Bell was a non-smoker.

Occupational and Environmental History:

34. Ms. Bell attended Central State Beauty College in Salisbury, North Carolina from approximately 1974-1975. She obtained her beautician's license in 1975. As part of her training, she worked with and around talcum powder, including Clubman talcum powder products. She applied the Clubman powder with a brush to the neck; more often this was used on men, but it was also used on women with short haircuts. She would put the powder onto her hand and then use the brush to apply it, or sometimes sprinkled the powder directly from the container onto the client's neck.

35. She worked for LaMarick Salon at Belk's Department Store in Salisbury, NC from around 1975-1978 where she was a hairdresser, working on male and female clients. She used Clubman talcum powder. Ms. Bell worked in her home as a hairdresser from around 1974 – 2009, in a business called Country Curls, when she converted her garage into a beauty salon. She continued to use Clubman talcum powder. Her clientele was around 50/50 male and female. She also cut children's hair and would use baby powder on the children under the age of two years, rather than Clubman talcum powder. Ms. Bell noted that she performed more haircuts on shorter hair than longer hair; she used the Clubman talcum powder for the shorter haircuts. Ms. Bell described the air as appearing "dusty" when she applied the talcum powder out of the container.

36. From 1967-1973 Ms. Bell worked for Fiber Industries as a beamer operator, working with polyester filaments and polymer yarns. She also worked in an area called draw twist. She worked for Cannon Mills for two quarters in 1969 and returned to work in the fourth quarter of 1976. She worked there until 1985, when it was changed to Fieldcrest Cannon, where she worked full-time until 1993. Ms. Bell worked with cotton fiber and was a weaver making sheets. She worked at Philip Morris from 1996-2009 as a packer.

37. Ms. Bell used Cashmere Bouquet talcum powder on a daily basis from the 1950s until the 1980s. In addition, she was exposed to dust from her mother and sisters' daily use of Cashmere Bouquet. Ms. Bell's daughter also used Cashmere Bouquet. Ms. Bell used Cashmere Bouquet from when she was 2 or 3 years old until the late 1980s. When she was young, she took a bath three to four times a week and her mother would apply the Cashmere Bouquet after the bath. When she was able to bathe herself, she applied the powder. Ms. Bell recalled that her mother used a powder puff to apply the powder to her body. She noted that when the powder was applied there would be a "puff of smoke and it went everywhere. It was on the sink and everywhere..." She applied powder to her younger sister for around two years as well after she assisted her sister with the bath, and was later present when her sister applied the powder herself. When she was 10, she began bathing daily and using Cashmere Bouquet after her bath. She cleaned up the bathroom after she used the powder with a rag for the counter, and a mop for the powder that was on the floor.

38. Ms. Bell's husband, Lloyd Bell, worked in spin draw and was a truck driver at Fiber Industry when they married in 1968. He worked as a driver for Propst Seafood starting in 1972, then worked fabricating aluminum window frames and then worked as a tractor trailer driver.

39. Ms. Bell's sister, Judy Drye, attended beauty school while Ms. Bell was in school as well. She noted that around 50% of the clients at the beauty school were male, and after they cut the men's hair, they would apply Clubman talcum powder. Ms. Drye recalled seeing her sister shake the Clubman talc onto a brush and apply it to the necks and faces of the clients after their haircut. She estimated that it took one to two minutes to brush the neck. She worked at Belks with Ms. Bell after completing cosmetology school. She recalled that both she and Ms. Bell used Clubman talcum powder on their clients. Ms. Drye also saw Clubman talcum powder at Ms. Bell's home salon. In their personal use, Ms. Drye noted that they used mostly Cashmere Bouquet after their bath, and the Cashmere Bouquet was applied with a powder puff. She noted that Avon products were used around 10% of the time, and Johnson and Johnson's Baby Powder was used if they ran out of Cashmere Bouquet.

40. Ms. Bell's sister Nancy Taylor recalled that her mother applied powder on the diaper and after the bath. She recalled that their mother used Cashmere Bouquet, Avon and Shower to Shower. Mr. Bell's brother Wayne Whitley noted that Ms. Bell used Clubman talcum powder after she cut his hair. She applied the powder to his neck with a brush.

41. Ms. Bell had malignant mesothelioma of the right chest as a result of her exposure to asbestos from cosmetic talc. She underwent two surgeries and chemotherapy. In addition, Ms. Bell had a hyaline pleural plaque, a hallmark finding for prior asbestos exposure that was found during the pathological review at the time of her thoracoscopy.

42. Based on the information available, it is my opinion, to a reasonable degree of medical certainty that Ms. Bell's exposure to asbestos-containing talcum powder led to the development of her mesothelioma. She began using Cashmere Bouquet talcum powder around 1950 and continued to use it until the 1980s. In addition, she used Clubman talcum powder starting in 1974 when she cut hair, and used it on a frequent basis in her work as a hairdresser.

Methodology

43. The methodology and basis for my opinions follows standard methods of the medical and scientific community.[11] Asbestos is the most well-known cause of mesothelioma, and the causation of mesothelioma has been established by the quantitative history of exposure to asbestos. Thousands of individuals, from myriad professions and exposure situations have developed mesothelioma as a result of either direct or indirect exposure to asbestos. The reliance on the history of exposure to asbestos was used by seminal studies by Newhouse, Wagner and Selikoff in the 1960s, who attributed mesothelioma to asbestos exposure based solely on the history of exposure. The increased risks for mesothelioma exist for individuals who both worked directly with asbestos products and for those who worked adjacent to or in the vicinity of others who were using asbestos products, which is known as "bystander" exposure.

[11] These methodologies are generally accepted in the scientific and medical community and have been described in the pier reviewed literature including by Bradford-Hill, Lemen, Freeman, and Welch.

Asbestos and Malignant Mesothelioma General Opinions

44. Occupational Medicine is the field of medicine that deals with exposures to substances, toxins, conditions and agents in the workplace that are associated with increased risks of diseases. It exists as a subspecialty of Preventive Medicine that deals with identifying ways to prevent people from becoming ill. This includes identifying the sources, agents or catalysts that increase the likelihood of someone developing a disease, illness, or detrimental condition, and educating people on how to eliminate, avoid, and/or mitigate those risks. To put it simply, Occupational Medicine and Preventive Medicine involves searching for and identifying causes of diseases. This knowledge is important for those who are already ill: elimination of the catalysts can eliminate or mitigate the illness. It is also important from a public health point of view: to a large extent, the higher purpose of Occupational Medicine and Preventive Medicine is to educate and warn the public on how to eliminate, avoid, or mitigate the risks of diseases at the workplace, and to provide guidance to governments and businesses on appropriate regulations and standards concerning workplace health and safety.

45. One of the essential tasks of a physician of Occupational Medicine, when dealing with an individual patient, is the taking of a proper occupational history. Standard medical histories usually involve the patient explaining their reason for seeking medical attention; a listing of current symptoms, conditions, allergies, medications and other relevant medical problems; and providing some family and social history. Occasionally, a standard medical history may – but does not always – include identifying the patient's occupation.

46. A full occupational history, on the other hand, will go into details of a patient's entire work history, including details concerning their tasks and duties and their working conditions and environment. The history will also routinely make inquiries into the patient's home or hobbies. It would also reveal what kinds of substances or agents the patient was exposed to in his or her working environment that might have occurred decades earlier. It remains the standard tool for determining exposure and has not been supplanted by quantitative measurements, which are rarely obtained, and would not, unless continuously performed on an individual (which is not feasible), fully address all exposures an individual might have had. At times, it is not possible to directly obtain an occupational history from an individual, and information concerning work and environmental experiences contained in deposition transcripts by plaintiffs, co-workers and family members can provide detailed information of that type that can be elicited from an occupational physician-obtained history.

47. The hallmark of occupational medicine is to connect an exposure to a hazardous substance to a disease, and identify whether there is a causal relationship. This is a critical differentiation in the field of occupational medicine; not only do we treat patients for disease, but we emphasis what hazardous substance might be causing the disease. In occupational medicine training, there are core areas of training, including epidemiology, biostatistics, toxicology, and industrial hygiene.

Asbestos and Disease:

48. Asbestos is a naturally occurring mineral that has been used commercially for a variety of purposes for over 100 years. Asbestos is mined in the form of microscopic fibers released

from the surrounding earth. Asbestos was extremely useful from an industrial perspective: it is highly resistant to heat and therefore serves as an excellent insulator and friction surface. It is also very durable, and as a fiber it can be molded into shapes and products that serve a variety of functions. However, asbestos is also highly toxic and carcinogenic when the fibers are inhaled or ingested.

49. While there are many "fiber types" of asbestos, as well as different sizes of the fibers, there exists consensus among scientists that exposure to any asbestos fiber type or size increases the likelihood of lung cancer, mesothelioma, as well as nonmalignant lung and pleural disorders. Asbestos fibers are generally divided into two categories: amphiboles and serpentine (or chrysotile). There are several varieties of amphiboles, including both commercial and non-commercial types. The three major asbestos types used in industry have been chrysotile, amosite and crocidolite. Of these three fiber types, over 95% of all asbestos used in the United States has been chrysotile. Much of the chrysotile asbestos that was used in the US was mined in Canada, where there was contamination with small amounts of tremolite, another type of amphibole asbestos. The mainstream scientific community has also long recognized, and continues to recognize today, that there is no "safe" level of exposure to asbestos regardless of fiber type or size. This position is shared by numerous United States government agencies, including the Occupational Safety and Health Administration ("OSHA", which has regulatory authority over workplaces), the Environmental Protection Agency ("EPA" which has regulatory authority over non-occupational settings), the National Institute for Occupational Safety and Health ("NIOSH", which is responsible for conducting research and making recommendations for the prevention of work-related injuries and illnesses), the World Trade Organization ("WTO"), and the national academies of science of every major industrialized nation. The World Health Organization recently reviewed the existing literature and concluded (in 2014) that all fiber types are capable of causing asbestos related disease, including mesothelioma, and reiterated the statement that there is no safe level for exposure to asbestos.

50. Due to the ubiquitous use of asbestos and its presence in naturally occurring formations, there is asbestos in the ambient air in the United States, albeit at minute levels. ATSDR reports ambient air concentration or "background level" to range from 0.0005 f/cc in urban areas, to 0.00005 f/cc in rural regions. EPA does not distinguish between urban and rural levels and reports background in the United States as 0.00001 f/cc. These levels are thousands of times less than the current OSHA permissible exposure level of 0.1 f/cc. While it is theoretically possible to develop mesothelioma from ambient air concentrations, it has not been proven to occur at levels at or below ambient air concentrations. Given that there is no truly "unexposed" population, it would be impossible to reasonably perform such a study to determine if this were the case.

State of the Art

51. In 1898 Montague Murray described interstitial fibrosis in an individual exposed to asbestos. Pancoast described radiographic changes of interstitial fibrosis in asbestos workers in 1917. Cooke described two cases of asbestosis in the 1920s, and actually used the term "asbestosis" to describe the interstitial fibrosis among asbestos workers, and also noted pleural plaques (fibrosis) in these workers.

52. In 1930 Merewether and Price, in their Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry, noted that inhaling dust containing asbestos fibers could lead to disabling and fatal lung disease. They studied asbestos workers in the textile

mills in Great Britain, and noted that asbestosis could occur in large numbers of exposed individuals. Moreover, they found that the textile workers with the highest exposures had more asbestosis than workers in areas where asbestos exposure was lower. Merewether and Price noted that asbestos was a potential hazard to health in any industry where dry asbestos products were abraded or otherwise manipulated to generate dust, such as thermal insulating. They recommended warning, education and training of all those individuals who were exposed to asbestos.

53. Lynch and Smith noted a case of lung cancer in an asbestos worker from South Carolina in 1935. Textbooks in the 1930s, such as A.J. Lanza's textbook on dust disease, included asbestosis as a disease of concern. In 1943, the first case of mesothelioma was associated with asbestos exposure and was published by Wedler in Germany. Also in 1943, Hueper from the United States Public Health Service stated that he believed asbestos caused lung cancer. He published an editorial stating this association in the Journal of the American Medical Association in 1949.

54. In 1955, Doll published a seminal article that described the increased risk of lung cancer among asbestos exposed workers. By the time of Doll's epidemiology study, there had been over 60 cases of asbestos-related lung cancer published in the literature. In 1960, Wagner et.al. published a study of 33 cases of malignant mesothelioma among individuals who were exposed to asbestos in and around the crocidolite mines in South Africa. Not only were miners developing disease, but family members, individuals on the wagon routes in which the asbestos was carried and people who had played with mine tailings as children developed mesothelioma. In the early 1960s numerous studies in several countries, under different exposure scenarios, were published that showed mesothelioma in association with asbestos exposure. In fact, by the end of 1964, over 700 scientific articles had been published that showed the adverse health effects of asbestos.

Development of Disease

55. When asbestos is inhaled, some proportion of the fibers can be deposited upon any component of the respiratory tract, including the nose, pharynx, conducting airways and the alveolar or gas exchanging regions of the lung. Fibers that land initially on the airways and above are cleared rapidly from the lung. The primary defense mechanism that mediated this clearance is known as the mucociliary escalator. The escalator is comprised of collated and mucous producing epithelial cells that propel inhaled fibers up to the mouth where they can be swallowed or expectorated. These epithelial lining cells are the "target cells" for cancers. Fibers that evade the mucociliary escalator can penetrate into the lower airways and lung tissue, where they can be transported through the body. Amphibole fibers tend to clear from the lung less rapidly than chrysotile fibers. Asbestos is cleared through the pulmonary lymphatics to lymph nodes and to the pleura, the target organ for pleural mesothelioma. Of the different fiber types, Suzuki, Sebastien and LeBouffant have all shown that chrysotile fibers preferentially translocate to the pleural space.

Asbestosis:

56. The fibers that are inhaled and deposited past the escalator can cause asbestosis. These fibers deposit initially on the Type 1 and Type 2 alveolar epithelial cells. On the epithelial surfaces, some asbestos fibers activate the 5th complement which attracts inflammatory cells, including foreign particles, like asbestos, from the lung. About 20% of the fibers deposited on the alveolar surfaces are enveloped by the Type 1 cells and are translocated to the underlying connective tissue (interstitial) compartment. There, the fibers can interact with interstitial fibroblasts,

myofibroblasts and macrophages. Fibroblasts and myofibroblasts are the target cells for asbestos because these are the cells that synthesize and release the scar tissue matrix. (See Y. Suzuki & N. Kohyama, Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues.,19 Am J. Indus, Med. 701 (1990); Y. Suzuki & N. Kohyama, Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues., 19 Am J. Indus, Med. 701 (1991)). They produce scar tissue when the epithelial cells are injured and when the macrophages are activated. Alveolar cells and macrophages release a number of protein growth factors that stimulate the fibroblasts to multiply and produce scar tissue and the fibroblasts and myofibroblasts also synthesize a similar array of factors that induce their own cell growth and matrix production that we recognize as asbestosis. Like all of the asbestos-related diseases, asbestosis is dose dependent. An individual typically needs long-term occupational exposure to develop clinical asbestosis.

57. The scarring process described above begins as soon as inhaled fibers are deposited on the alveolar surfaces, and microscopic asbestosis is ongoing in the lungs of afflicted individuals for many years before any clinical signs or symptoms are presented. The initial physiological symptom of asbestosis is shortness of breath. This is caused by the scar tissue which replaces normal elastic connective tissue, this producing a stiff lung that restricts the individual from taking a deep breath. Shortness of breath also results when scar tissue thickens the alveolar-capillary membrane, the barrier across which oxygen and carbon dioxide gases are exchanged.

Pleural Plaques and Fibrosis:

58. This is scar tissue formation in an identical manner to that described above, under asbestosis. The difference is that there is little direct deposition of asbestos fibers in the pleura. While some fibers can be inhaled through the alveolar ducts and reach the pleura directly, most fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to the pleura do so by way of pulmonary lymphatic flow. The inhaled fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to lymphatic fluids which flow through these regions on the way to the pleura. The lymphatic flow carries fibers to the pleura where they interact with the sub-mesothelial fibroblasts that produce a scar tissue matrix, as described above. If the scarring is in a circumscribed pattern, the scarring is called "plaque". Investigators have shown that this injury can result in a restrictive lung disease in some individuals.

Lung Cancer:

59. These tumors caused by asbestos typically arise in cigarette smokers, although some epidemiologic studies on asbestos-exposed non-smokers show an increased risk of developing the disease. When an individual is exposed to the cancer-causing agents (carcinogens) of both cigarettes and asbestos, the risk of getting lung cancer is increased well beyond the risk presented by exposure to either agent alone or by simply adding the risks of the two carcinogens. Epidemiologists multiply the risks of the two carcinogens since there is a clear synergy in the way asbestos and cigarette smoke combine to cause lung cancer.

60. Cancer is the loss of control of cell growth. Every cell in the bodies of humans and animals is under strict genetic control of the rate at which a given cell replaces itself by dividing. Cancer is caused when the specific genes that control cell division and other aspects of the cell cycle develop errors or mutations. Carcinogens induce such errors, and complete carcinogens can produce the errors with no other agent required. Cigarette smoke has a number of complete carcinogens, and

all of the asbestos varieties have been shown to act as complete carcinogens. Thus, as the airway epithelial cells of the mucociliary escalator are assaulted daily by cigarette smoke and asbestos fibers, a number of cells are injured, and many exhibit genetic errors through the lifespan of the individual. In those who are susceptible to developing a cancer, one of those injured cells accumulates a sufficient number of genetic errors in genes that control cell growth to finally, after decades of exposure, lose the normal growth pattern and grow into a malignant tumor. (See Frost G, Darton A, Harding AH. The effect of smoking on the risk of lung cancer mortality for asbestos workers in Great Britain (1971-2005) Ann Occup Hyg 55:239-24 (2011)).

Mesothelioma:

61. This cancer occurs when a mesothelial cell of the pleural, peritoneal, or pericardial surfaces develops a sufficient number of genetic errors in a set of genes that control cell growth, as described above. Cigarette smoking has no influence on the development of mesothelioma. (See N.S. Offermans, et. al., Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherland Cohort Study, 56 J. Occupational Envt'l Med. 1 (2014); Robinson BM. Malignant pleural mesothelioma: an epidemiological perspective, 1 Annals Cardiothoracic Surgery 491 (2012)).

62. Asbestos exposure is the only known occupational and/or environmental cause of mesothelioma in North America, and all of the asbestos varieties induce the genetic errors described above and cause this cancer. The fibers that cause mesothelioma reach the pleural surfaces through the lymphatic pathways, as explained earlier, and they interact with the target cells of the mesothelial surfaces. When a sufficient number of genetic errors have accumulated in a single mesothelial cell, this cell can undergo neoplastic transformation and grow into a deadly tumor. It typically takes many decades for a sufficient number of mutations to occur in a single mesothelial cell because of the numerous effective defense mechanisms that destroy genetically defective cells, thus explaining the long latencies known for this cancer.

63. All of the asbestos varieties have been shown to cause genetic errors and fibers less than five microns can bind DNA and this contributes to the development of genetic damage. Short fibers have been found to accumulate in the pleural regions of the lung as well as in mesenteric lymph nodes of the peritoneal cavity. Longer fibers may be comparatively more dangerous than short fibers (on a fiber per fiber basis), but all size ranges are capable of causing and contributing to the development of mesothelioma or any of the asbestos-related diseases. Exposure to asbestos fibers of all types and lengths are toxic, and short fibers more readily reach the mesothelial target cells of the pleura. (See Y. Suzuki & S. R. Yeun, Asbestos Fibers Contributing to the Induction of Human malignant mesothelioma., 982 Annals N.Y. Acad. Sci. (2002); Y. Suzuki, et al. Short thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence., 208 Int'l. J. Hygiene Env. Health 201 (2005)).

64. Some have suggested that geological nomenclature – calling the anthophyllite and tremolite in the talc either "non-asbestiform" or "cleavage fragments" – has biological significance. This notion has been rejected by the EPA, US Centers for Disease Control and Prevention, Agency for Toxic Substances and Disease Registry, USGS (United States Geological Survey), the American Thoracic Society, and most recently by the FDA Working Group, and is not a distinction that is considered medically important. In fact, mesotheliomas have been documented among New York State miners and millers of talc containing approximately 50% "non-asbestiform" anthophyllite

and tremolite. Asbestos related diseases have also been found at the Vermont talc mines and mills. The absence of documented cases of mesothelioma among one cohort of miners and millers of talc containing less than 1% the tremolite and anthophyllite (such as the Italian studies of talc miners and millers) is most likely due to an inadequate sample size, selection criteria, and the manner in which the data has been reported. (US EPA Region 9 Response to the 2005 National Stone, Sand and Gravel Association Report, April 20, 2006; RT Vanderbilt Co., MSDS, May 1, 1975; Roggli, et.al. Tremolite and Mesothelioma., Ann Occ Hyg 46(5):447-453 (2002); Lamm, Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers; Epidemiology-Fibers, 1576-1581 (1988); Exhibit E, June 26, 2019 Report of Dr. Madigan). Recently, Mirabelli noted a case of mesothelioma in a 79 year old man who was a maintenance worker who worked at the Italian talc mill from 1947-1957 (Mirabelli D, Letter on: "Cosmetic talc as a risk factor for pleural mesothelioma: a weight of evidence evaluation of the epidemiology, Inhal Toxicol 29(8):341 (2017). A mesothelioma in Johnson & Johnson's consumer product division worker population has been reported.[12] This individual's exposure in the consumer product division would be more analogous to what a consumer of talcum powder products would experience than a miner or miller's exposure. Johnson & Johnson's own internal records document many dozen mesotheliomas with Johnson & Johnson associating the possible cause to be exposure to its talcum powder products.[13]

65. Fibers of all lengths can bind to DNA and cause genetic errors that are required in the causation of cancer such as mesothelioma. Fiber burden studies of mesothelioma patients show a preponderance of chrysotile asbestos within the tumor tissue. Since the target location of mesothelioma is the pleura, the lung burden of asbestos does not reflect the fact that asbestos has moved from the lung to the pleura, where it can cause the mesothelioma to develop. (See Ronald F. Dodson, Analysis and Relevance of Asbestos Burden in Tissue, in Asbestos: Risk Assessment, Epidemiology and Health Effects. Risk Assessment, Epidemiology and Health Effects 78 (2d, ed. 2011); M. Silverstein, et al., Developments in Asbestos Cancer Risk Assessment. Am J. of Indus. Med. (2009)).

66. Moreover, there is ample evidence to support the conclusion that exposure to chrysotile asbestos fibers-typically used in brake linings-can and does cause mesothelioma. This conclusion is supported by, among others, the American Conference of Governmental Industrial Hygienists, the American Thoracic Society, the Environmental Protection Agency, the International Agency for Research on Cancer, the National Toxicology Program, OSHA, the Consumer Products Safety Commission, the World Health Organization, and the World Trade Organization. The scientific consensus that all fiber types and sizes can cause mesothelioma is also reflected in the Consensus Report of the 1997 Helsinki Conference (discussed below) and publications from the American Cancer Society and the National Cancer Institute of the National Institutes of Health.

67. In essence, there exists a consensus among the overwhelming majority of medical and scientific professionals and organizations that asbestos fibers of any type or size can cause mesothelioma, including chrysotile fibers. (See Dodson, Ronald F. et al., Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review, 44 Am J. Indus. Med. 291 (2003); D.

[12] Protected Documents (workers compensation records) produced by Johnson & Johnson, Bates Number JNJTALC000534203-53690.

[13] Protected Documents (Adverse Event Reports) produced by Johnson & Johnson, varying bates numbers.

Egilman, et al., Exposing the "Myth" of ABC, "Anything But Chrysotile: A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies. Am J. Indus. Med. 540 (2003); David S. Egilman & Marion Billings: Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability, 11 Int. J. Occupational Envtl Health 360 (2005). 11:360-371; Egilman D. Fiber Types, Asbestos Potency, and Environmental Causation. 15 Int. J. Occupational Envtl. Health (2009); Finkelstein, M. Asbestos Fiber Concentrations in the Lungs of Brake Workers: Another Look, Annals Occupational Hygiene 455 (2008); M.M. Finkelstein & C. Meisenkothen, Malignant Mesothelioma among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos. 54 Annals Occupational Hygiene 692 (2010); P.J. Landrigan, et al., The Hazards of Chrysotile Asbestos, a Critical Review. 37 Indus. Health 271 (1999); W.J. Nicholson,The Carcinogenicity of Chrysotile Asbestos-A Review. 39 Indus. Health 57 (2001); R.A. Lemen, Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model. 10 (2) Int. J. Occupational Envtl. Health (2004); see also R. Lemen, Asbestos in Brakes: Exposure and Risk of Disease. 45 Am. J. Indus. Med 229 (2004); EPA: Guidance For Preventing Asbestos Disease Among Auto Mechanics. (1986); A.H. Smith & C.C. Wright, Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma. 30 Am. J. Indus. Med. 252 (1996); U.S. Dept. of Labor: Working Safely with Asbestos in Clutch and Brake Linings. (posting); U.S. Dept. of Labor, OSHA Directorate of Science, Technology and Medicine, Office of Science and Technology Assessment. Asbestos-Automotive Brake and Clutch Repair Work; World Health Organization, Environmental Health Criteria 203: Chrysotile Asbestos. International Programme on Chemical Safety (1998 Geneva)).

68. Asbestos fibers are very small; so small, in fact, that millions of fibers could fill the air in a room without anyone being able to perceive it with the naked eye. The fibers are odorless, cannot be seen with the naked eye, and are aerodynamic. Consequently, someone can inhale asbestos fibers without even being aware of it. The fibers are also small enough to pass through the normal respiratory defense mechanisms that the human body uses to keep out toxins and debris.

69. The Scientific community has even concluded that small amount of asbestos exposure can cause cancer. The Rodelsperger study indicates that exposure to asbestos below the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) of 0.1 fibers per cubic centimeter can cause disease. However, visible asbestos-laden dust that is released into the air from the manipulation of gaskets or packing, or that is reintroduced into the respirable zone from the process of sweeping the floor, is between 2.0 and 10.0 fibers per cubic centimeter. These levels far exceed the OSHA PEL. Some of these levels even exceed the OSHA PEL issued in 1972.

70. Government agencies and international organizations universally recognize asbestos as a carcinogen in low levels. These agencies include the International Agency for Research on Cancer, Environmental Protection Agency, OSHA, National Institute for Occupational Safety and Health, and World Health Organization. The inhalation of asbestos fibers also does not trigger any immediate physiological reactions: the victim doesn't experience any immediate irritation, asthmatic problems, or allergic reactions. Moreover, the latency, or development period, for mesothelioma is very long: the minimum latency period is usually considered to be around 10 years from first exposure with a maximal latency period well over 60 years after the last exposure. Consequently, it could be decades before someone is aware that he or she was exposed to asbestos, or it might have occurred so remotely that they do not realize they had asbestos exposure. Moreover,

they may not realize that a product they used contained asbestos and thus are unaware they had exposure.

The Helsinki Criteria for Attribution:

71. In January 1997, a conference called "Asbestos, Asbestosis and Cancer" was held in Helsinki, Finland. The conference was convened to establish criteria for diagnosis and attribution of disorders of the lungs and pleura, including mesothelioma. This was a multidisciplinary group of internationally recognized experts, consisting of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the members had published over 1,000 articles on asbestos and associated disorders. The conclusions of the conference were developed into a peer- reviewed Consensus Report that established the "Helsinki Criterion". Among the conclusions of the Helsinki Criterion are:

a. That, in general, reliable work histories provide the most practical and useful measures of occupational asbestos exposure; and
b. That even in the absence of other independent evidence of disease (e.g. lung fiber counts exceeding the background range for the lab in question; the presence of radiographic or pathological evidence of asbestos-related tissue injury; histopathologic evidence of abnormal asbestos content), a history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution of the disease with asbestos exposure.

Moreover, with reference to determining an occupational etiology of mesothelioma, the Helsinki Criterion Consensus Report concluded that:

a. The great majority of mesotheliomas are due to asbestos exposure;
b. Mesothelioma can occur in cases with low asbestos exposures. However, very low background environmental exposures carry only an extremely low risk;
c. About 80% of mesothelioma patients have had some sort of occupational exposure to asbestos (necessitating a carefully obtained and detailed occupational history for proper diagnosis);
d. An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related;
e. A minimum of 10 years from the first exposure is required to attribute mesothelioma to asbestos exposure (though in most cases, the latency interval is longer);
f. Smoking has no influence on the risk of mesothelioma.

72. The conclusions of the Helsinki Criterion have since been adopted by, and form the general consensus of, the medical community's position vis-à-vis mesothelioma and asbestos. (See *Consensus Report, Asbestos, asbestosis and cancer: the Helsinki criteria for diagnosis and attribution*, 23 Scandinavian J. Work Environ Health 311 (1997)). And, given the fact that about 80% of patients with mesothelioma have had some sort of occupational exposure to asbestos,[14]

[14] The remaining 20% of mesothelioma patient likely had asbestos exposures that were para-occupational or are simply unidentified.

asbestos exposure in the workplace is a prime focus of Occupational Medicine when dealing with mesothelioma patients.

Mesothelioma Is A Dose Responsive Disease:

73. It is my opinion that Mesothelioma and asbestos related lung cancer are dose responsive diseases in which more substantial exposures directly increases the risk for the development of these cancers. This linear dose-response relationship presented in Asbestiform Fibers: Non-occupational Health Risks, published by the National Research Council National Academy of Sciences in 1984, discussed herein, is neither new nor novel and generally accepted in the medical and scientific communities. As per the aforementioned Helsinki criteria, the first question usually asked of a patient diagnosed with mesothelioma, concerns how, when, and where the patient was exposed to asbestos. (*See* Consensus Report, Asbestos asbestosis and cancer: The Helsinki criteria for diagnosis and attribution. 23 Scandinavian J. Work Environ Health 311 (1997)). Because of the proven association between asbestos fibers and mesothelioma, proof of significant exposure to asbestos dust is considered to be proof of specific causation. (*See* P. Boffetta, et al., Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment. 89 (6) Medicina Del Lavoro, 471 (1998). This causal relationship between exposure to asbestos dust and the development of mesothelioma is so firmly established in the scientific literature that it is accepted as a scientific "fact".

74. Malignant mesothelioma is, in general, a dose response disease where one's cumulative exposure to asbestos-containing dust has been shown to contribute to cause diffuse malignant mesothelioma including pleural mesothelioma. (*See also* Newman, et al., Malignant Mesothelioma Register 1987-1999. 74 Int'l Arch Env. Health 383 (2001), (concluding that "higher cumulative asbestos-fiber dose leads to the earlier development of mesothelioma)). As each exposure to asbestos contributes to the total amount of asbestos that is inhaled, and, in doing so, reduces the necessary period for asbestos disease to develop. Therefore, each non-trivial exposure to asbestos should be considered a contributing factor in the development of malignant mesothelioma or lung cancer. More recently, the BAP-1 gene mutation has been found to confer increased susceptibility in individuals who have both the mutation and have asbestos exposure. There is no evidence that the genetic mutation of this tumor suppressor gene without asbestos exposure cases mesothelioma.

Exposure to Asbestos Contaminated Talc and Disease

75. Asbestos fibers have been reported in cosmetic talcum powder for decades, in company documents, the media, FDA communications, and the published medical and scientific literature. In 1935 asbestos was identified as a source of exposure in talc miners and millers by Dreesen. By 1968 Cralley had described asbestos in consumer cosmetic talc products. By 1972, the cosmetic industry was looking for asbestos free alternatives to cosmetic talc.

76. Cosmetic talc has been analyzed by researchers in various countries, and has routinely been shown to contain asbestos. In 1957, researchers at Battelle Memorial reported finding tremolite in Italian talc used by Johnson & Johnson, the same Italian talc used in Cashmere Bouquet and Clubman talc. In 1968, Johns-Manville documented fibrous tremolite asbestos in consumer cosmetic talcum powder products, including Cashmere Bouquet. In 1972, Snider, et al., reported finding asbestos in several consumer cosmetic talcum powder products, including Johnson's Baby

Powder and Cashmere Bouquet. That same year, Lewin of New York University reported finding asbestos in Johnson's Baby Powder and Cashmere Bouquet purchased off the shelf. In 1973, Lewin further found asbestos in Clubman talc purchased off the shelf. The University of Minnesota also found asbestos in Lewin's sample of Johnson's Baby Powder. In 1974 Rohl, and in 1976, Rohl and Langer tested 20 consumer products that had been labeled as talc or talcum powder, including body powders. Of the 20 products that were tested, ten were found to contain tremolite and anthophyllite, principally asbestiform. Of note, the product that had the highest asbestos content in the Rohl and Langer study was Cashmere Bouquet, the same product later tested by Gordon, et. al. Pooley, while consulting for Johnson & Johnson, also found anthophyllite in the Cashmere Bouquet Rohl and Langer studied, confirming the finding. In 1981, Churg and Warnock proposed that cosmetic talc was the source of the finding of tremolite/anthophyllite in women's lung tissue. Similarly, in 1989, Roggli found tremolite/anthophyllite in women's lung tissue and hypothesized the source to be cosmetic talc. In 1997, McDonald attributed the finding of tremolite in lung tissue of a chrysotile worker to his prior work exposure to talc occupationally as a barber. Mattenklott et. al. in 2007 found that small amounts of talcum powder (0.1 gram) released significant amounts asbestos fibers. In 2015, Ilgren, et al. attributed the increased rate of mesothelioma in the chrysotile miners in Italy to the tremolite asbestos in the talc in the adjacent mining region.

77. Exposure to asbestos-containing talc has been shown to cause asbestos related diseases, including mesothelioma. A recent paper by Gordon, et. al., *Asbestos in Commercial Cosmetic Talcum Powder as a Cause of Mesothelioma in Women*, evaluated the mineralogical constituents of a consumer talcum powder product – Cashmere Bouquet – and its ability to release asbestos fibers into the breathing zone of the direct user and bystanders. In their paper Gordon et. al. noted that the talc that was used in Cashmere Bouquet was derived from three distinct regions where anthophyllite and tremolite asbestos were found. Gordon et. al. measured 18 million anthophyllite asbestos fibers per gram in the talcum powder. Air measurements were done by both phase contrast microscopy ("PCM") and transmission electron microscopy ("TEM"), and significant levels of asbestos fibers were noted (anthophyllite, tremolite and some chrysotile) in the breathing zone of the individual applying the powder as well as a bystander. Results taken from the experiment in the paper show that personal measurements from the shaker container test showed a measurement by PCM of 4.8 f/cc, with an actual asbestos fiber measurement of 1.8 f/cc. Bystander measurements showed a lower, but still significant exposure of 1.35 f/cc by PCM for the bystander, and 0.5 f/cc of actual asbestos fibers. Similar measurements were done with the puff application method. Personal measurements after using a puff were 23.6 f/cc and 16.5 f/cc for the user, with actual asbestos fiber measurements of 5 f/cc and 3.5 f/cc. A short term sample showed even higher measurements, of 60 f/cc with the use of a puff and actual asbestos fiber measurements of 13 f/cc. Bystander exposures to asbestos from the puff application were elevated, with a short term sample by PCM of 13.7 f/cc and 9.7 f/cc, and an actual asbestos fiber measurement of 4.9 f/cc and 3.5 f/cc. Gordon et. al. also noted that the TEM measurements were far more sensitive than x-ray diffraction detection, since there was a much lower detection limit with TEM.

78. In addition, the Mine Safety and Health Administration ("MSHA") monitored personnel in the mill where Italian talc was ground (this talc was used in consumer products) in 1984. The filters from the personal measurements from these workers contained 5.8% anthophyllite. The report issued determined that this equated to anthophyllite comprising 0.6% of the bulk Italian

talc.[15] Recently, Dr. Steven Compton found asbestos in 11 of the 13 samples collected from the Italian mines, from which the talc originated that was then used in consumer products – including Cashmere Bouquet and Clubman – including anthophyllite, tremolite, actinolite, and chrysotile. His findings are consistent with the findings documented in the historic literature from testing done contemporaneously to Betty Bell's product use and exposure, including testing done by Johns-Manville, commercial laboratories hired by companies including Avon, Johnson & Johnson and Colgate-Palmolive, and talc suppliers like Cyprus/Imerys and Whittaker Clark & Daniels, Professor Lewin of New York University, and others.

79. Further, the recent testing by Dr. William Longo of Cashmere Bouquet from vintages during Betty Bell's product use and exposure and Dr. Steven Compton and Sean Fitzgerald's testing of Ms. Bell's own Clubman and other Clubman products from vintages during Ms. Bell's use and exposure found tremolite and anthophyllite asbestos in these products. (Exhibit B, Exhibit C, Exhibit D) Dr. Longo analyzed and reported on approximately 58 Cashmere Bouquet products that were manufactured from 1910 through 1977 and provided to his laboratory from mesothelioma plaintiffs, from collectors, and from Colgate-Palmolive's own historic collection (20 obtained directly from Colgate-Palmolive). (Exhibit B) These products encompass the eras of all three talc sources for Cashmere Bouquet talc products offered for sale in the United States (Italy, North Carolina, Montana) although Colgate-Palmolive used Italian talc in every container of Cashmere Bouquet it manufactured in the United States.[16] His testing has shown regulated asbestos in containers sourced from each of these mines and decades. These findings are consistent with Colgate-Palmolive's own analysis of the Cashmere Bouquet and talc ore sources during the years Ms. Bell used and was exposed to those products.[17] Dr. Longo report regulated asbestos in 51 of the 58 (88%) containers of Cashmere Bouquet talcum powder products. (Exhibit B)

80. Three independent laboratories have identified asbestos in Betty Bell's own container of Clubman.[18] Mr. Fitzgerald of SAI, Inc. and Dr. Compton of MVA both identified tremolite asbestos in the Clubman container Ms. Bell found in her home and gave to her lawyers. (Exhibit C, Exhibit D) Mr. Fitzgerald also performed a product use simulation with Ms. Bell's Clubman container, applying it to a model head following the application description provided by Ms. Bell during her deposition testimony, using her barber brush. (Exhibit D) Four low-volume air samples and eight high-volume air samples were taken in the simulated tests. All four simulations produced quantifiable airborne asbestos, and all revealed quantifiable tremolite asbestos by TEM analysis. (Exhibit D) The average aspect ratio of the tremolite fibers identified by SAI was 15:1 with the vast majority of the fibers greater than 5 microns in length. Dr. Compton has also identified anthophyllite asbestos in three containers of Clubman as well as a barber brush containing Clubman talc that were owned by a hairdresser named Margaret Lashley. (Exhibit C)[19]

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[15] May 15, 1984 – Report of MSHA visit to Cyprus Industrial Minerals Company's South Plainfield Mill.

[16] Deposition of Michael Burke, Colgate-Palmolive Corporate Representative, taken in *Allen v. Colgate-Palmolive Company, et al.*

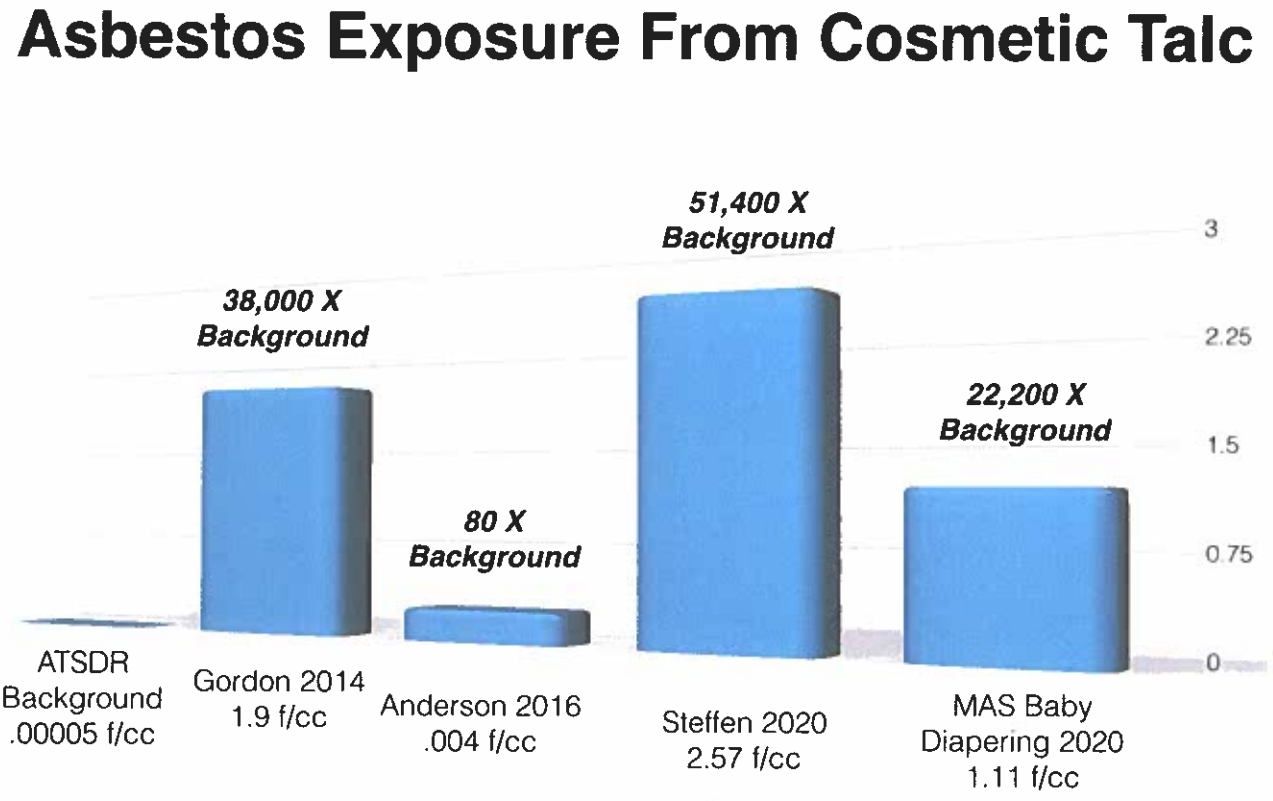
[17] A true and correct copy of the Testing of Colgate's Cashmere Bouquet Summary is attached hereto as **Exhibit F**.

[18] SAI, Inc. of Greensboro, North Carolina, Lab/Cor of Seattle, Washington, and MVA Scientific Consultants of Duluth, Georgia.

[19] Lawyers for AII have speculated during my depositions that the asbestos identified in Ms. Bell and Ms. Lashley's Clubman containers – stored sealed in their homes – came from the ambient air in North Carolina.

81. Dr. Longo's laboratory has performed additional analysis to determine whether exposure from use of talcum powder below the waist (using Italian talc-sourced Johnson's Baby Powder), led to respirable levels of asbestos. Samples taken in the breathing zone of an individual during use of Johnson's Baby Powder below the waist, consistent with the manner in which Betty Bell was exposed, resulted in a mean tremolite fiber exposure of 2.57 fibers/cc. Area samples taken resulted in a mean tremolite fiber exposure of 0.2 fibers/cc. (Exhibit B) Dr. Longo's study was published in the peer-reviewed scientific literature in 2020.[20]

82. Exposure data from talcum powder usage as compared to background is identified on the graph below:



The epidemiological literature consistently shows a substantially increased risk for disease at these cumulative levels of exposure.[21]

Not only is there no actual evidence of such a contamination, but such a contamination would be nearly impossible given that the tiny holes in the tops of the containers of Clubman were sealed. (*See* testimony of Ms. Bell and Ms. Lashley as well as photographs taken of the products upon finding). Further, Mr. Fitzgerald conducted air monitoring inside his laboratory as well as of the outside air in Greensboro, North Carolina, to compare to the levels he identified in the Clubman product. (Exhibit D) Mr. Fitzgerald reported that the baseline ambient values determined inside and outside of his laboratory were below detectable levels inside the laboratory (less than the analytical sensitivity of 0.00002 asbestos structures/cc), and found right at the detectable levels in testing outside the building at an ambient average level of 0.00005 structures/cc.

[20] Steffen, BA, et al., *Serous Ovarian Cancer Caused by Exposure to Asbestos and Fibrous Talc in Cosmetic Talc Powders—A Case Series*, JOEM, Vol. 62, No. 2 (February 2020).

[21] *Iwatsubo*: OR 4.2 for >0.5 -0.99 f/cc-yrs (95% CI 2.0-8.8); *Rodelsperger*: OR 20.5 for >0.15-1.5 f/cc-yrs, cumulative exposure ending 20+ years before diagnosis (95% CI 5.8-72.6); *Lacourt*: (women) OR 23.7 for

83. In addition to looking at bulk and air samples, Gordon et. al analyzed the lung tissue and lymph node tissue of a woman who had been exposed to contaminated talcum powder (Cashmere Bouquet). The authors found that there were 3150 and 4150 fibers per gram wet weight, respectively, with a detection limit of 690 fibers per gram wet weight. All fibers were 5 micrometers or greater in length, and had an aspect ratio of 20:1 or greater. The fibers were identified as anthophyllite or tremolite. In addition to the fibers counted above, there were many anthophyllite and tremolite fibers that were less than 5 micrometers in length, with a predominance of anthophyllite. In the lymph node, amphibole asbestos fibers were also noted, measuring 12,738 fibers per gram wet weight (detection limit 2123 fibers per gram wet weight). Again, the fibers noted were anthophyllite and tremolite. In addition to the asbestos found in the lungs, the authors noted fibrous and platy talc and small asbestos bodies.

84. The issue of asbestos and talc has been studied for decades. Millman 1947 noted pneumoconiosis in a man exposed to cosmetic talc. Lung scarring was seen in miners from New York State in the 1950s, and there are elevated rates of mesothelioma and lung cancer in miners at the asbestos contaminated talc mines. Moskowitz 1970 reported a talc pneumoconiosis in a woman exposed to cosmetic talc while working as a quality control inspector on the production floor at Revlon for 11 years. The International Agency for Research on Cancer has noted that talc containing asbestos is carcinogenic. More recently, case reports were published out of Italy involving patients with mesothelioma and exposure only to cosmetic talcum powder.[22] In 2019, my co-authors and I published a case series of 33 mesotheliomas with talcum powder usage as the only source of asbestos exposure for all 33 cases. Tissue digestion for six of the 33 cases was described in detail. Uniformly, the tissue fiber burdens revealed the presence of talc and asbestos fibers (anthophyllite, tremolite, and/or chrysotile) typical of the contaminants found in cosmetic talcum powder.[23] In 2020, Emory, et al. published a case series of 75 mesotheliomas with talcum powder usage as the only source of asbestos exposure for all 75 cases, including a selection of tissue fiber burden analysis revealing the presence of talc and asbestos fibers.

Applying an Accepted Method for Evaluating Disease Causation in an Individual

85. In deciding whether Ms. Bell's mesothelioma was caused by her exposure to asbestos, I applied the methodology that was described by Welch, et.al. in her paper *Asbestos Exposure Causes Mesothelioma, but Not This Asbestos Exposure: An Amicus Brief to the Michigan*

>0.1 (99% CI 3.3-168.53).

[22] Andrion, Alberto, et al. *Malignant Peritoneal Mesothelioma in a 17-Year-Old Boy with Evidence of Previous Exposure to Chrysotile and Tremolite Asbestos*, Human Pathology, Volume 25, No. 6 (June 1994); Musti, et al. *Exposure to Asbestos and Mesothelioma Risk of Onset of Primary Ovarian Description of Two Cases*, JNJ 000383914-18. [Musti, et al. reported a case of malignant biphasic mesothelioma in a 23 year old woman with no known history of asbestos exposure professionally, environmentally, or from her family. The authors noted there was substantial use of talc for hygiene from birth up to 5-6 years of age. Tissue digestion and fiber burden analysis identified tremolite and talc. The authors explained this correlation by noting it "is established that the talc especially in the past was sometimes contaminated with asbestos fibers in virture of the common mineralogical origin."]

[23] Moline, Jacqueline, et al., *Mesothelioma Associated with the Use of Cosmetic Talc*, Journal of Occupational and Environmental Medicine, 2020.

Supreme Court, published in 2007 in the International Journal of Occupational and Environmental Health.[24] In this paper, she identifies four questions that should be examined in the causation of disease in an individual:

1. Was the individual exposed to a toxic agent?
2. Does the agent cause the disease present in the individual?
3. Was the individual exposed to this substance at a level where the disease has occurred in other settings?
4. Have other competing explanations for the disease been excluded?

86. For question #2, there is ample literature that asbestos causes mesothelioma and no dispute in the medical literature. With respect to question #1, Ms. Bell had an exposure to asbestos from Cashmere Bouquet and Clubman talc for many decades, fulfilling this criterion. Cashmere Bouquet and Clubman talc have been shown to contain asbestos and Ms. Bell would have had asbestos exposure based on her and her family's description from their deposition testimony. Even if Ms. Bell had exposure from her employment or her household member's employment before she was a hairdresser, my opinion with regard to exposure from Cashmere Bouquet and Clubman talc would not change. Assuming that the facilities Ms. Bell and her household members worked at did contain friable asbestos to which Ms. Bell was actually exposed, any exposure that Ms. Bell sustained from these products would have contributed to her overall dose along with the Cashmere Bouquet and Clubman talc; they are in addition to (and do not negate) her substantial exposure to asbestos from Cashmere Bouquet and Clubman talc (#4).[25] The remaining criterion, #3 is whether there is an analogous exposure scenario in which others also developed mesothelioma. As described above, and recently referenced by the Center for Disease Control as well as published in the peer-reviewed literature,[19] there are numerous other individuals with exposure to asbestos-containing talc products who have developed malignant mesothelioma.[26]

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[24] The methodology is consistent with the published methods of Lemen and Freeman, and is generally accepted in the medical community.

[25] A numerical dose calculation is not required by any government agency or the medical community in order to attribute an individual's mesothelioma to asbestos exposure. The Helsinki Criteria, described above, along with Welch, Lemen, and Freeman, provide the basis for attribution of an individual's mesothelioma to asbestos exposure without having to calculate a specific numerical dose. Qualitative descriptions of exposure to respirable asbestos-containing dust are sufficient for attribution. Calculating an individual's *actual* "dose" from a specific product is a theoretical exercise unless air data was collected contemporaneously for that individual's exposures. Betty Bell did not wear a dust collector and there were no filters mounted in her home or place of work collecting dust during her use of and exposure to Cashmere Bouquet and Clubman talc. As a result, a true dose can never be calculated.

[26] Andrion, Alberto, et al. *Malignant Peritoneal Mesothelioma in a 17-Year-Old Boy with Evidence of Previous Exposure to Chrysotile and Tremolite Asbestos*, Human Pathology, Volume 25, No. 6 (June 1994); Bulbulyan, M.A., et al., *Cancer Mortality Among Women in the Russian Printing Industry*, AM J Ind Med, 36:166-171(1999); Finkelstein, M., *Malignant Mesothelioma Incidence Among Talc Miners and Millers in New York State*, Am J Indust Med 55, 863-868 (2012); Ghio, A, Roggli, V, *Talc Should Not Be Used for Pleurodesis in Patients with Nonmalignant Pleural Effusions*, Am J Respir Crit Care Med, Vol 164, No. 9, pp 1741 (2001); Fujiwara, Hiroshi, et al. *An Autopsy Case of Primary Pericardial Mesothelioma in Arc Cutter Exposed to Asbestos through Talc Pencils*, 43 Industrial Health 346-350 (2005); Ilgren E, et al., *Critical reappraisal of Balangero chrysotile and mesothelioma risk*, Epidemiology Biostatistics and Public Health, Vol. 12, No. 1 (2015); Lamm, S.H., et al., *Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers*, Epidemiology-Fibers, 1576-1581, (1988); Mirabelli

Conclusion

87. Based on the information that was provided to me, and applying both my understanding of the medical literature and the facts of this case, it is my opinion to a reasonable degree of medical certainty that the exposures to the dust from asbestos-containing Cashmere Bouquet and Clubman talc that Betty Bell used and was exposed to for nearly five decades, starting in the mid-1950s, were above normal background levels and that her use of and exposure to asbestos from Cashmere Bouquet and Clubman talc was a substantial contributing factor in her development of mesothelioma. Ms. Bell's exposure to asbestos was the cause of her mesothelioma and premature death.

I declare under the penalty of perjury that the foregoing is true and correct.

Executed on June 30, 2020 at New York, New York.

JACQUELINE MOLINE, MD, MSc, FACP, FACOEM

D, *Letter on: "Cosmetic talc as a risk factor for pleural mesothelioma: a weight of evidence evaluation of the epidemiology"*, Inhalation Toxicology, 29:8, 341 (2017); Musti, et al., *Exposure to Asbestos and Mesothelioma Risk of Onset of Primary Ovarian, Description of Two Cases*, (2009); Moline, Jacqueline, et al., *Mesothelioma Associated with the Use of Cosmetic Talc*, Journal of Occupational and Environmental Medicine, (2020); Emory, Theresa, et al., *Malignant mesothelioma following repeated exposures to cosmetic talc: A case series of 75 patients*, Am J Ind Med, (2020).